PHYSIOLOGICAL EFFECTS OF VARIATION IN LEVELS OF RESPIRED CARBON DIOXIDE AND OXYGEN IN THE CHICKEN

by

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INTRODUCTION

The field of respiratory physiology is slowly yielding its secrets. Advancements being made in the study of the effect of pH, P_{CO_2}, and P_{O_2} interaction on respiration, in the study of peripheral chemoreceptor functions, and in the study of the mechanical properties of the lung are making possible a clearer understanding of respiratory function.

Of the peripheral chemoreceptors which have been isolated, the carotid body has been studied in the most detail (Joels and Neil, 1963; Comroe, 1964). Whether or not the peripheral chemoreceptors all function in the same way and whether or not there is interaction of responses to pH, P_{CO_2}, and P_{O_2} within a given peripheral chemoreceptor, or possibly even between the various peripheral chemoreceptors, has yet to be determined.

In studying the respiratory response to the stimulation of a single, isolated chemoreceptor it is necessary to control the blood gas tensions reaching all other chemoreceptors. In general, arterial blood gas tensions are very nearly the same as alveolar gas tensions. Thus, by controlling the gas tensions at the exchange surfaces of the lung, it is possible to produce desired tensions of these gases in the blood.

Although respiratory and circulatory responses to variations in oxygen and carbon dioxide levels have been studied in man, only isolated observations have been made for the chicken. This paper presents a method by which arterial P_{CO_2} and P_{O_2} can be predicted, within a given range, for the chicken when the gas mixture being given through the unidirectional respirator is known. In conjunction with the determination of arterial blood gas tensions and pH, a study of the respiratory and circulatory responses to variations in respiratory gas tensions in the chicken has also been made.

LITERATURE REVIEW

In studies of peripheral chemoreceptors, some attention has been given to the control of arterial blood gas levels. MacLeod and Scott (1964) simply estimated arterial oxygen tension from alveolar oxygen tension in the study of the carotid body. Duke et al. (1963) bubbled 5 percent CO_2 in oxygen through the blood of the "restricted" systemic circulation in order to keep it constant in their study of the effects of stimulation of the pulmonary body. Daly et al. (1965) used mechanical oxygenation of the blood while studying the aortic body.

The study of respiratory physiology in the chicken and the experimental control of blood gas levels has been aided by the development of a unidirectional respirator (Burger and Lorenz, 1960). Using this respirator a gas mixture can be introduced through a tracheal cannula, forced through the lungs and into the air sacs, and allowed to exit via an opening produced surgically through the midventral abdominal wall and the abdominal air sacs. The unidirectional respirator was modified (Fedde and Burger, 1962) to allow the gas to be heated and humidified, thus preventing lowering of the body temperature and dehydration of the pulmonary tissue. Use of this respirator allows open thoracic surgery in the chicken under conditions which provide a responsive experimental subject for many hours.

Arterial Blood Gas Tensions

Control of arterial blood gas tensions should be possible using the unidirectional respirator. Arterial gas tensions have been found to be quite close to alveolar gas tensions. In man (normal) an alveolar PO_2 of 97.4 mm Hg corresponds to an arterial P_{O_2} of 97.1 mm Hg, according to the results of Comroe and Dripps (1947). During normal respiration the amount of oxygen in the inspired air is diluted by the CO_2 and water vapor in the lungs, so that the alveolar air has a lower oxygen tension than inspired air. With the use of the unidirectional respirator, the dilution process should be virtually eliminated and the arterial gas levels should be under more exact experimental control; thus, a certain mixture of gases introduced through the respirator should correspond to certain arterial gas tensions, which can be determined and controlled.

The oxygen dissociation curves, which have been determined for the chicken, provide information which helps in the interpretation of blood gas tensions (Christensen and Dill, 1935). Morgan and Chichester (1935), in their determination of oxygen dissociation curves for the chicken, found that blood taken from a resting chicken had an arterial P_{O_2} of 90 mm Hg and P_{CO_2} of 34 mm Hg at a temperature of 40.0° C. In general, these dissociation curves show that chicken blood has less affinity for oxygen than the blood of man.

Respiratory and Circulatory Effects

Studies of respiratory and circulatory responses to variations in oxygen and carbon dioxide tensions in respired gases have provided some interesting results from mammals, especially man. In a review of the circulatory effects of hypoxia, Korner (1959) points out that there is an increased heart rate and cardiac output and that peripheral vasoconstriction occurs. Some investigators have not found an increase in cardiac output during hypoxia (Glick et al. 1964). Thilenius et al. (1964), in support of active vasoconstriction of the pulmonary vessels during hypoxia, found that 6-15% O_2 produced an increase in pulmonary artery pressure and cardiac output and a decrease in left atrial pressure. From these values the pulmonary vascular resistance was calculated and found to increase.

In a study of effects of increasing the CO_2 in inspired gas, Schneider and Truesdel (1922) found that increases in blood pressure, heart rate, and respiratory minute volume occurred in man. In a review article on the control of respiration, Kellogg (1964) states that Pflüger, in 1868, did the first experimental work to show that hypercapnia and hypoxia stimulate breathing. Kellogg shows that for man, increasing CO_2 increases respiratory minute volume, tidal volume, and rate, and decreasing O_2 increases ventilation (liter/min.). He points out that the ventilatory responses to O_2 and CO_2 do not appear to be simply additive. Increasing the CO_2 increases the response to hypoxia, and decreasing the O_2 increases the response to hypercapnia. According to Kellogg, the response for an increase in H^+ appears to be additive to the response for an increase in carbon dioxide, as Gray (1950) originally proposed. However, Hamilton (1964) (using a high constant PO_2) found that CO_2 and H^+ interact negatively with an increase in one producing a decrease in response to the other.

Little is known concerning the effects of such gaseous changes for the chicken. Hiestand and Randall (1942) have shown that 10 per cent CO_2 in an unanesthetized chicken will increase the respiratory amplitude. Van Matre (1957) ventilated chickens by introducing respiratory gases through a needle inserted into the posterior thoracic air sac. He found that a rather large flow of gas would inhibit respiration, but that increasing the amount of N_2 or CO_2 would overcome the inhibition. Increasing the CO_2 increased the depth of respiration, although the rate of respiration did not appear to change.

MATERIAL AND METHODS

The experiment was divided into two studies. Ten adult Single Comb White Leghorn males were used in the first study, while fourteen Hy-Line[1] males were used in the second study. Each study was further divided into two parts. In Study I, Part A, the oxygen in the dry gas mixture of the "inspired" gas was held constant at 18%, while the carbon dioxide was varied from 20-0-20%. In Part B, the carbon dioxide was held constant at 5%, while the oxygen was varied from 4-94%. In Study II, Part A, the oxygen was held constant at 20% of the dry gas mixture, while carbon dioxide was varied from 12-2-12%; in Part B, carbon dioxide was held constant at 5%, while the oxygen varied from 50-6% in two trials (1 and 2).

There were a few differences in experimental design between Study I and Study II. In Study I, Part A, carbon dioxide levels in the respiratory gas ranged from very low (0%) to rather high (20%). The higher carbon dioxide levels (above 12%) and the low carbon dioxide levels (below 2%) were not used in Study II, mainly because of possible deleterious effects on the bird of higher levels of CO_2 and because the pCO_2 electrode responds logarithmically and, hence, cannot accurately be used to measure these low levels. The 50% to 94% mixtures of oxygen were not used in Study II, Part B, because little change in any parameter measured (except arterial P_{O_2}) occurred at these levels. In Study II, the bird first received 50% O_2; then the O_2 was gradually reduced to the lower levels. In Study I, Part B, the oxygen mixtures were initially dropped to 4% O_2 and gradually raised in steps to 94% O_2.

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[1] Coombes and Sons Hatchery, Sedgewick, Kansas.

An Offner Type S Dynograph[2] was used for the simultaneous recording of arterial blood pH, arterial blood P_{O_2}, arterial blood P_{CO_2}, arterial blood pressure, tracheal pressure, and sternal movements. Body temperature was monitored throughout the experiment using a read-out thermometer[3] with a rectal thermistor[4], which was inserted four inches into the rectum.

Experimental Materials and Arrangement

The unidirectional respirator. The unidirectional respirator and other parts of the experimental arrangement are presented diagrammatically in Fig. 1. The unidirectional artificial respirator, as described by Burger and Lorenz (1960) and Fedde and Burger (1962), is divided into the flowmeter section and the heater-humidifier section. Four floating-ball flowmeters[5] were used for regulating and measuring the flow of air, oxygen, nitrogen, and carbon dioxide. Although calibration curves were provided by the manufacturers of the flowmeters, determination of these curves was repeated for the specific gases used over the required range of flowmeter settings. This was done in the following manner. At each flowmeter setting, a soap bubble was introduced into the flow of gas passing from the flowmeters into a graduated glass tube. The gas flow carried the bubble through the graduated glass tube (10 ml.), and the movement of the bubble was timed to obtain the flow (ml./min.). A larger tube (200 ml.) was used for the faster flows at higher settings of the flowmeters, since the rapid flow in the small tube was too fast for accurate timing. The tube was

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[2] Offner Division of Beckman Instruments, Inc., 3900 River Road, Schiller Park, Illinois.

[3] Yellow Springs Instrument Co., Yellow Springs, Ohio (Model 43).

[4] Yellow Springs Instrument Co., Yellow Springs, Ohio (Model 401).

[5] Precision Laboratory Instruments, Cole-Parmer Instrument and Equipment Co., 7330 North Clark Street, Chicago 26, Illinois.

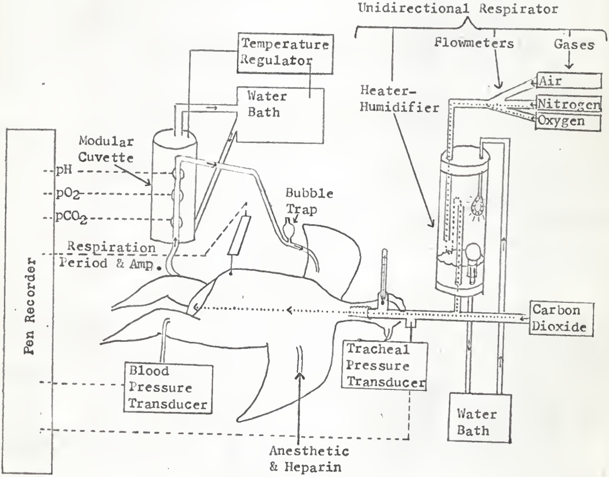

Fig. 1. Experimental arrangement for the simultaneous recording of arterial blood pH, arterial blood P_{O_2}, arterial blood P_{CO_2}, arterial blood pressure, tracheal pressure and sternal movements during alterations in respiratory P_{O_2} and P_{CO_2}.

rinsed with water periodically to prevent drying and breaking of soap bubbles. The new calibration curves showed some slight differences in gas flow over the range used when compared with the original calibration curves (Fig.2).

The gas heater-humidifier section (Fig. 1) of the unidirectional respirator consisted of a large, stoppered, glass tube which contained a fine spray of warm water. The water was obtained from a constant temperature water bath (± 0.5 °C.) and was continuously recirculated. Air, or oxygen-nitrogen mixture, from the flowmeters was introduced into the heater-humidifier through a tube which reached almost down to the water level. The gases then passed up through the spray of water to the outlet tube, which passed down and out of the heater-humidifier. Since carbon dioxide is very soluble in water, it was added to the gas mixture as the mixture passed from the heater-humidifier before reaching the tracheal cannula. The temperature of the gas mixture, as it passed into the trachea, was held at 42°-44° C. by controlling the temperature of the water bath. This procedure helped to maintain the body temperature of the bird at about 40° C. The heater-humidifier thus provided saturation of the gases with water vapor and reduced loss of moisture and heat from the lungs. The gases were thus considered to have been warmed and humidified to within the normal limits for gases in the lungs. Gross examination of the lungs after an experiment showed them to be normal in color and density.

The pO_2, pCO_2, and pH electrodes. For measurement of arterial P_{O_2}, P_{CO_2}, and pH the Beckman modular cuvette with electrodes[6] was used. This device provided a continuous extracorporeal blood gas sensing system. The electrodes were the oxygen macro electrode, the Severinghaus pCO_2 electrode, the silver-silver chloride micro blood pH electrode, and the fiber junction reference

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[6] Spinco Division, Beckman Instruments, Inc., Stanford Industrial Park, Palo Alto, California.

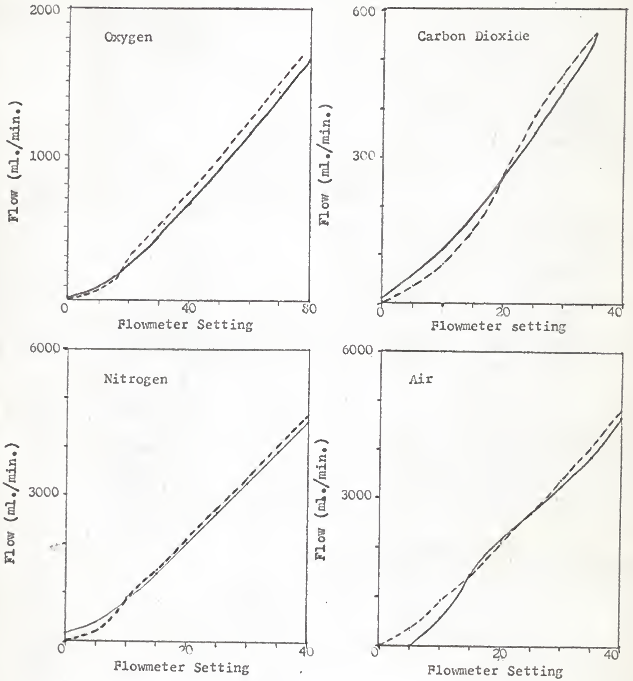

Fig. 2. Comparison of flowmeter calibration curves using the soap bubble technique with manufacturer's calibration curves. New curve —— ; from manufacturer's curve--- .

electrode.

Since the values measured with the electrodes vary with the temperature, it was necessary to control accurately the temperature in the cuvette. A custom-made water bath incorporating a temperature controller,[7] an immersible thermistor,[8] a heater[9] (200 watts), and a vertical immersion recirculating pump[10] was used with the modular cuvette to hold the temperature of the water circulating through the cuvette at 40.00° ± 0.07° C. Severinghaus (1964) has provided correction factors for the effect on P_{O_2} of variation of temperature and pH in human blood. However, these factors are not applicable for the chicken, since the blood pH and the oxygen dissociation curves for the chicken are different from those of man.

The pO_2 and pCO_2 electrodes were also used as a means of determining the accuracy of the oxygen and carbon dioxide tensions in the gas mixture at the various flowmeter settings which were presented to the birds. The expected oxygen tensions for the gas mixtures at body temperature and atmospheric pressure saturated with water vapor (BTPS) were calculated for the various gas percentages according to the equation: P_{O_2} = per cent oxygen (barometric pressure - water vapor pressure at 40° C.). For example, at 10 per cent oxygen, $P_{O_2} = .10$ (727.9mm Hg - 54.9 mm Hg) = 67.3 mm Hg. The expected tensions were compared with those obtained when the gas mixtures produced by given settings of the flowmeters were measured with the electrodes. A comparison of the values for P_{O_2} is presented in Fig. 3. There was close agreement between expected and measured values, although measured values were slightly lower at the upper oxygen tensions. Figure 4 shows the close agreement between the expected and measured values of P_{CO_2}. In general, the comparisons indicated that, at a desired flowmeter setting, the bird was receiving close to the expected gas tension.

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[7] Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio (Model 71).

[8] Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio (Number 403).

[9] Aloe Scientific, St. Louis, Missouri.

[10] Aloe Scientific, St. Louis, Missouri.

Calibration of the oxygen and carbon dioxide electrodes required the use of calibrating gases.[11] Certified analysis of these gases showed purity to be 5.031% CO_2 and 12.26% CO_2. Nitrogen (99.999%) was used for zero oxygen. Air was used as a standard for 20.9 per cent oxygen. In initial experiments, the calibrating gases were passed quite slowly and intermittently through the cuvette in order to minimize cooling and drying of the electrodes. In later experiments the calibrating gases were first bubbled through warm water in a test tube in order to help warm and humidify the gas before it reached the electrodes. This procedure increased the repeatability of the calibrations.

The pH electrode was calibrated using buffers with values of 6.84, 7.38, and 7.84 at 40° C.[12]

Statham pressure transducers[13] were used to sense blood pressure and tracheal pressure. A mercury manometer was used for calibration of the blood pressure transducer and a water manometer was used for calibration of the tracheal pressure transducer. Tracheal pressure was not measured in the first

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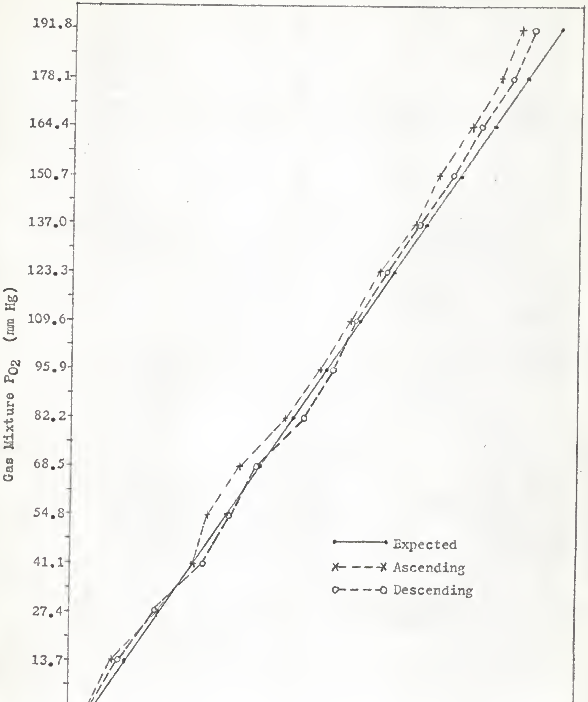
[11] Air Products of Minnesota, Inc., P.O. Box 176, Shakopee, Minnesota.

[12] Spinco Division, Beckman Instruments, Inc., Stanford Industrial Park, Palo Alto, California.

[13] Statham Laboratories, Inc., Hato Rey Industrial Subdivision, Hato Rey, Puerto Rico. Blood pressure transducer (Model P23Gb), tracheal pressure transducer (Model P23AA).



P_{O_2} with oxygen electrode

Fig. 3. Comparison of expected P_{O_2} in the gas mixtures from the flowmeters with the P_{O_2} obtained when the gas mixtures were analyzed with the pO_2 electrode. Both descending and ascending changes were made in the gas mixtures. The obtained P_{O_2} was slightly lower than expected at the higher tensions. At the average barometric pressure during the study 13.7 mm Hg = 2% O_2.

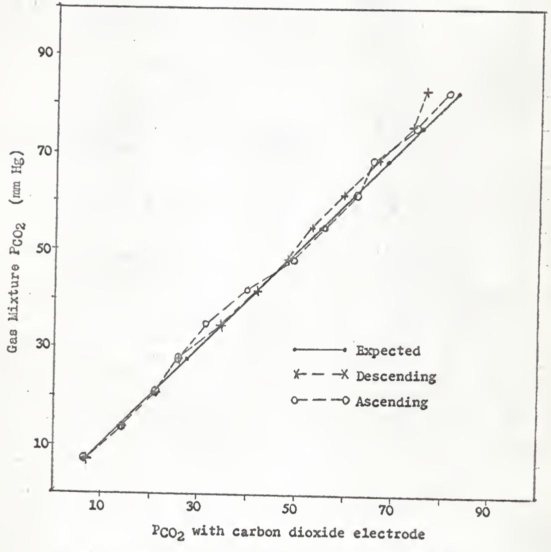


Fig. 4. Comparison of expected P_{CO_2} in the gas mixtures from the flowmeters with the P_{CO_2} obtained when the gas mixtures were analyzed with the pCO_2 electrode. Both descending and ascending changes were made in the gas mixtures. At the average barometric pressure during the study 13.7 mm Hg = 2% CO_2.

study; but it was included in the second to obtain an indication of any change in the resistance to air flow presented by the lungs. Tracheal pressure was taken a few centimeters from the tracheal cannula (Fig. 1).

Respiratory movements were measured with a strain gauge device, which was attached near the caudal tip of the sternal carina (Fedde et al., 1963). Respiratory amplitude and period (seconds/respiratory cycle) were measured.

Surgical and Experimental Procedure

The birds were restrained in dorsal recumbency and a polyethylene cannula (PE 90, 10 cm. in length) was placed in the left cutaneous ulnar vein for the administration of anesthetic and heparin. Pentobarbital sodium (65 mg./ml.) was infused slowly to effect. Absence of a response to a pinch on the rostral edge of the comb indicated deep anesthesia (Fedde et al., 1963). Usually 1.0 to 1.5 ml. of anesthetic were required. Additional 0.1 ml. doses were given as needed throughout the experiment to maintain a light surgical anesthetic level. Cannulae for blood pressure measurement (PE 160, 27 cm. in length) and for provision of a flow of arterial blood (PE 160, 20 cm.) to the extracorporeal blood gas sensing system were placed in the left and right ischiatic arteries respectively.

Blood was returned to the bird from the extracorporeal blood gas sensing system by inserting a cannula (PE 120, 32 cm.) into the right cutaneous ulnar vein. A one-piece, glass bubble trap (Fig. 1) was inserted in the path of the cannula a few centimeters from the bird. The bubble trap was essentially a perpendicular extension from a small glass tube (through which the blood flowed) forming a bulb which had an opening at the top into which was inserted a small serum bottle stopper. A small air bubble could be injected into the three-way stopcock between the cuvette and the return cannula in order to measure the flow of blood. When the bubble reached the trap, it rose into the bulb and thus did not enter the vascular system of the bird. The trapped air could occasionally be removed from the bulb by a syringe and needle inserted through the rubber serum bottle stopper in the bubble trap. The movement of the air bubble along the marked length of the cannula was timed in order to provide an indication of the rate of flow of the blood through the cuvette. Satisfactory flows were on the order of 0.15 ml./sec. At this flow rate, it required less than four seconds for blood to reach the sensing device from the bird.

With the cannulae in place, preparation was made for the flow of the gases through the bird. A skin incision (5 cm. in length) was made midventrally in the proximal half of the neck. Two layers of connective tissue were transected and the trachea (on the right side of the neck) was isolated in preparation for later tracheotomy. A midventral abdominal incision was made from the caudal tip of the sternal carina to within 1 cm. of the cloacal orifice, and the abdominal air sacs were ruptured. The tracheotomy was then completed, and a mixture of air and 5% carbon dioxide from the unidirectional respirator was introduced through the tracheal cannula. The gas mixture passed through the lungs, into the air sacs, and out to the atmosphere through the openings in the air sacs and the abdominal wall. The cranial and caudal thoracic air sacs were then ruptured using an alligator biopsy forceps. The cervical air sacs had been ruptured during the installation of the tracheal cannula. Thus, gas could pass over almost all of the respiratory gas exchange surface. In the interest of economy, a gas mixture of air and 5 per cent carbon dioxide was used until the beginning of the experimental series at which time oxygen and nitrogen were substituted for air. At all times, a total flow of 4000 ml./min. of gas was given to the bird. Four to six per cent carbon dioxide had been found to give normal respiratory movements for the anesthetized chicken under unidirectional, artificial respiration (Fedde et al., 1963). This level of carbon dioxide prevents the drastic changes in acid-base balance which would occur if only air were used. Heparin sodium[14] (initial dose of 200 units) was infused slowly over a 10 minute period with additional 10 unit doses given during the experiment about every 30 minutes. The heparin was used in order to prevent clotting in the cannulae and, especially, to prevent clotting in the modular cuvette.

The flowmeters were then set to give the desired flows of the gases. For each change in the gas mixture, a time of at least three to five minutes was allowed for the change in the recorded value of the arterial gas tensions to stabilize before any readings were taken. For example, with a change from 7% CO_2 + 20% O_2 + 73% N_2 to 8% CO_2 + 20% O_2 + 72% N_2, readings for recorded values were taken after the change in the recorded arterial P_{CO_2} had leveled off. A continuous record was taken.

The measured values for each of the parameters (except for sternal movements and heart rate) were transformed from millimeters of recorder pen deflection to the appropriate units using the calibration curves which had been obtained. For every change in respired gas tension, the mean of each parameter (blood pressure, heart rate, arterial P_{CO_2}, etc.) was found over all birds. In a few cases not all parameters could be recorded. The standard errors of the means were calculated.

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[14] Abbott Laboratories, North Chicago, Illinois (Panheparin^R).

RESULTS AND DISCUSSION

Tables 1 through 5 present the numerical results of Study I and Study II. The results are graphically summarized in Figs. 5 through 16. The figures for each part are divided into a graph correlating changes in respiratory gas tensions with blood PO_2, PCO_2, and pH and a graph correlating changes in these gas tensions with blood pressure, heart rate, respiratory movements, and, in Study II, tracheal pressure. The respiratory gas tensions used are based upon the average barometric pressure for Study I and the average barometric pressure for Study II.

Effect of Variation in Respiratory PCO_2 with Constant PO_2 on Arterial $PCO2$, PO_2, and pH

The changes found in arterial PCO_2, PO_2, and pH with variation of respiratory carbon dioxide tension are shown in Fig. 5 for Study I and in Fig. 6 for Study II. The arterial PCO_2 was found to be in close agreement with the PCO_2 of the respired gas PCO_2, except for a slight difference at the low carbon dioxide tensions. Figure 7 provides a more direct comparison of the $PCO2$ values. The differences at the low carbon dioxide tensions were from a few mm. Hg in Study II to about 10 mm. Hg in Study I. The main differences in Study I started at about 10% CO_2 (descending) to about 5% CO_2 (ascending), with the largest differences at the low carbon dioxide tensions.

There are several possible explanations for this difference. First, the PCO_2 electrode response is logarithmic and not as rapid at the low tension of carbon dioxide. Due to the slow response of this electrode (1-2 minutes) a long time may be required for an accurate reading, especially below about 2%.

Table 1. Experimental values for variation in carbon dioxide in Study I.

Changes in CO_2 [3]		Changes in parameters (mean ± S.E.)							
% CO_2	Ave. P_{CO_2} mm Hg [1]	P_{CO_2}	P_{O_2}	pH	Sys. Blood Pressure	Dias. Blood Pressure	Heart Rate	Resp. Period	Resp. Amplitude
		mm Hg	mm Hg		mm Hg	mm Hg	Beats/min	cycles/sec	units[2]
20	136.4	134.0 ± 2.4	136.3 ± 6.4	7.11 ± .02	152.8 ± 8.5	104.2 ± 7.4	301.0 ± 5.3	2.9 ± .2	7.0 ± .7
19	129.6	130.4 1.9	136.9 6.6	7.12 .00	152.6 11.0	104.3 8.5	305.4 5.4	2.8 .2	7.0 .7
18	123.1	123.9 1.8	136.6 6.5	7.12 .02	149.6 10.5	102.1 8.7	306.6 4.8	2.6 .2	6.9 .7
17	116.0	115.0 1.4	136.3 6.1	7.15 .03	147.0 9.9	117.1 9.6	310.4 5.6	2.6 .3	6.7 .7
16	109.2	109.2 .6	134.8 5.6	7.15 .02	147.2 9.2	98.7 8.6	309.5 4.3	2.6 .2	6.5 .6
15	102.3	103.1 .7	132.7 6.1	7.19 .02	144.3 9.2	98.2 8.3	307.0 5.0	2.5 .2	6.2 .6
14	95.5	96.9 1.4	129.0 9.2	7.22 .02	142.7 8.8	98.2 7.8	301.6 5.3	2.4 .2	6.0 .6
13	88.7	89.4 1.7	129.3 8.1	7.26 .02	139.8 8.2	95.3 7.7	302.2 5.3	2.5 .2	5.6 .6
12	81.9	85.0 1.8	127.6 7.9	7.28 .02	137.6 7.3	93.6 7.2	294.4 5.6	2.4 .2	5.6 .6
11	75.0	78.5 1.7	126.5 8.2	7.32 .02	136.9 7.8	92.5 7.6	288.6 6.7	2.5 .2	5.2 .6
10	68.2	76.8 3.1	125.8 7.9	7.35 .02	134.7 7.8	89.0 7.6	287.0 6.5	2.5 .2	4.9 .5
9	61.4	66.4 1.6	123.1 8.5	7.38 .02	134.4 7.4	89.7 7.2	284.5 9.6	2.4 .2	4.4 .5
8	54.6	61.5 1.6	118.9 9.0	7.42 .02	131.2 7.4	88.5 7.3	279.4 8.6	2.4 .2	4.1 .5
7	47.8	58.2 2.1	116.5 8.8	7.44 .02	128.8 7.3	85.6 7.6	279.5 7.8	2.3 .2	3.6 .5
6	40.9	51.3 2.4	113.1 8.2	7.45 .06	125.8 6.6	81.6 6.5	274.6 9.2	2.2 .3	2.6 .4
5	34.1	44.9 2.4	111.6 9.3	7.51 .04	122.2 6.0	73.7 7.2	270.9 11.5	2.5 .6	1.4 .4
4	27.3	38.5 2.1	105.2 9.1	7.60 .04	113.8 8.4	66.2 7.9	248.4 14.4	3.7 .6	1.0 .1
3	20.5	33.0 2.3	101.9 9.1	7.64 .05	107.9 10.0	62.2 7.4	257.2 13.0	7.4 .6	.5 .1
2	13.6	27.4 2.1	99.1 9.8	7.68 .04	107.2 8.8	56.3 8.1	256.4 14.1	.9 .0	.6 .0
1	6.8	18.3 1.0	87.4 11.4	7.80 .01	105.6 8.6	59.9 4.5	256.3 15.4	.0 .0	.0 .0
0	0	12.4 .9	85.6 15.0	7.90 .05	103.2 9.4	56.5 7.5	248.0 18.6	.8 .0	.4 .0
1	6.8	22.3 1.4	92.9 10.0	7.76 .06	107.4 9.1	56.5 7.5	269.5 15.6	.0 .0	.0 .0
2	13.6	21.8 1.1	92.7 11.4	7.72 .03	110.0 8.1	59.6 7.1	270.8 15.7	.0 .0	.0 .0
3	20.5	27.2 1.2	93.9 11.8	7.72 .05	125.4 6.6	74.8 6.4	281.2 14.8	.2 .0	.3 .0
4	27.3	31.7 1.4	97.3 11.9	7.62 .03	125.8 6.3	77.9 6.7	281.6 14.4	1.6 .4	1.2 .3
5	34.1	37.6 1.6	101.1 11.9	7.63 .06	127.0 6.1	81.1 7.6	287.4 13.0	2.3 .4	2.2 .6
6	40.9	43.5 2.1	105.7 12.0	7.52 .04	131.6 6.6	85.2 7.8	288.0 12.9	2.4 .3	2.5 .6
7	47.8	49.1 2.5	108.6 12.2	7.49 .04	133.0 7.4	87.3 7.5	295.2 11.6	2.4 .3	2.9 .4

Table 1. (cont.)

Changes in CO_2[3]		Changes in parameters (mean ± S.E.)							
% CO_2	Ave. P_{CO_2} mm Hg[1]	P_{CO_2}	P_{O_2}	pH	Sys. Blood Pressure	Dias. Blood Pressure	Heart Rate	Resp. Period	Resp. Amplitude
		mm Hg	mm Hg		mm Hg	mm Hg	beats/min	cycles/sec	units[2]
8	54.6	54.1±3.0	112.1±12.1	7.46±.04	131.6 ±6.6	86.6± 7.2	316.8±14.7	2.3±.2	4.3± .5
9	61.4	59.4 3.4	112.0 11.9	7.42 .00	135.3 8.5	86.4 6.2	319.0 11.3	2.2 .2	4.9 .5
10	68.2	66.7 4.1	112.2 11.5	7.40 .04	136.3 6.3	87.5 5.3	314.4 11.8	2.4 .3	5.5 .4
11	75.0	72.1 4.6	114.0 11.6	7.36 .00	138.8 6.7	92.2 6.2	316.7 11.4	2.3 .2	5.9 .5
12	81.9	80.4 2.9	116.1 11.7	7.36 .00	138.9 6.3	93.8 6.5	314.6 10.8	2.3 .2	6.3 .5
13	88.7	87.5 2.7	115.0 11.9	7.28 .00	143.5 7.1	97.0 6.8	311.0 11.1	2.4 .2	6.7 .6
14	95.5	96.0 2.5	116.6 11.9	7.24 .00	141.5 7.2	96.6 7.7	319.0 9.5	2.5 .3	6.9 .7
15	102.3	103.3 2.9	118.1 12.2	7.21 .00	147.0 7.8	100.0 13.8	325.8 8.5	2.4 .0	7.0 .6
16	109.2	110.0 3.7	117.4 12.6	7.20 .01	149.2 8.7	103.6 9.9	331.3 9.3	2.4 .3	7.1 .6
17	116.0	117.7 3.1	119.7 12.6	7.17 .02	152.0 9.9	105.8 10.5	322.0 7.9	2.4 .3	7.2 .7
18	123.1	128.8 3.2	121.8 12.5	7.12 .01	151.6 10.0	108.6 10.5	321.0 6.8	2.4 .3	7.3 .3
19	129.6	135.4 2.5	123.4 12.2	7.10 .00	155.4 10.5	112.6 11.2	320.7 6.8	2.4 .3	7.4 .6
20	136.4	142.3 3.3	127.4 12.6	7.09 .01	158.3 12.1	111.9 11.6	318.7 6.4	2.4 .3	7.1 .6

[1] Based on average barometric pressure of 737.1 mm Hg.

[2] 1 unit = 5 mm pen deflection

[3] Changes in respiratory carbon dioxide with constant oxygen.

Table 2. Experimental values for variation of oxygen in Study I.

%O2 Changes [3]		Changes in parameters (mean ± S.E.)							
% O_2	Ave.[1] P_{O_2} mm Hg	P_{CO_2}	P_{O_2}	pH	Sys. Blood Pressure	Dias. Blood Pressure	Heart Rate	Resp. Period	Resp. Amplitude
		mm Hg	mm Hg		mm Hg	mm Hg	beats/min	cycles/sec	units[2]
4	27.3	44.3±1.0	53.8± 6.6	7.53±.01	113.3±11.3	57.5±11.8	320.8± 8.4	1.7±.1	5.0±.7
6	63.4	45.5 .9	63.4 7.8	7.52 .02	140.9 9.4	63.2 11.0	337.7 4.4	1.6 .1	5.3 .7
8	77.8	46.4 1.2	77.8 7.4	7.52 .02	142.2 9.4	73.6 10.7	344.4 5.4	1.7 .1	5.5 .7
10	86.2	47.0 1.2	86.2 8.7	7.51 .02	142.2 8.0	77.2 11.0	321.8 11.8	1.8 .1	5.3 .8
12	96.4	47.6 1.2	96.4 6.0	7.50 .02	140.2 8.1	79.4 11.2	330.3 10.5	1.9 .2	5.4 .7
14	103.6	47.7 .6	103.6 8.8	7.49 .03	139.2 8.6	80.9 10.6	329.6 9.8	1.9 .1	5.5 .7
16	111.7	46.9 .6	111.7 9.6	7.51 .02	138.1 8.4	79.4 9.8	323.2 16.9	2.0 .2	5.4 .7
18	116.9	48.4 1.2	116.9 12.6	7.51 .03	137.9 8.9	83.5 10.5	321.1 12.7	2.1 .1	5.3 .7
20	134.5	46.1 .7	134.5 12.6	7.52 .03	137.0 9.0	82.2 10.6	320.3 14.1	2.1 .2	4.9 .7
22	143.0	45.9 .7	143.0 13.5	7.52 .03	135.7 8.9	81.0 11.0	316.7 12.9	2.1 .2	5.2 .7
24	144.5	46.0 .5	149.5 12.4	7.53 .04	137.4 8.8	83.2 6.9	315.5 15.0	2.2 .1	5.3 .7
26	155.9	46.2 .2	155.9 13.3	7.53 .04	137.9 9.0	84.2 10.8	317.9 14.4	2.1 .2	5.3 .6
28	162.8	45.3 .9	162.8 13.2	7.52 .05	137.2 8.9	80.9 11.0	306.7 15.9	2.0 .2	5.0 .7
30	165.0	46.8 .8	165.0 15.0	7.51 .05	136.4 9.2	81.9 11.0	317.6 16.7	2.0 .3	5.2 .7
34	176.9	46.5 1.0	176.9 17.9	7.50 .04	138.2 8.7	83.3 10.5	314.0 16.5	2.0 .3	5.4 .7
38	192.4	46.6 1.2	192.4 18.0	7.51 .04	137.6 8.7	83.7 10.4	313.6 16.1	2.1 .2	5.6 .7
42	210.2	46.7 1.5	210.2 18.0	7.52 .04	138.5 8.7	83.3 10.7	309.0 17.5	2.0 .2	5.4 .7
46	221.2	47.4 1.0	221.2 22.5	7.51 .04	137.0 8.5	83.9 11.2	308.5 17.3	2.1 .2	5.4 .8
50	244.3	47.8 3.3	244.3 18.6	7.54 .05	138.2 9.1	84.7 10.6	309.8 18.3	2.1 .2	5.5 .8
56	382.0	47.0 .8	268.4 22.8	7.52 .04	140.7 4.4	86.6 11.7	309.1 18.2	2.0 .1	5.3 .8
62	423.0	46.9 1.0	299.0 26.6	7.51 .04	137.2 8.8	82.8 10.3	306.8 18.3	2.1 .2	5.4 .8
68	463.9	45.9 .9	336.8 28.0	7.53 .04	137.7 8.9	83.3 11.1	307.4 17.9	2.0 .2	5.0 .8
74	504.8	45.8 1.1	382.2 27.4	7.53 .05	130.4 9.7	87.2 11.8	302.8 17.4	2.0 .2	4.9 .7
80	545.8	45.4 1.0	409.3 25.0	7.53 .06	136.0 8.8	82.9 11.7	316.9 20.0	1.9 .2	4.6 .7
86	586.7	45.1 1.1	441.4 32.0	7.53 .04	134.6 8.7	83.5 10.7	317.4 20.2	1.9 .2	4.6 .7
92	627.6	45.0 1.4	454.5 40.7	7.53 .05	134.2 8.1	84.9 10.4	319.8 19.8	1.9 .3	4.1 .8
94	641.3	45.4 1.2	491.3 45.0	7.52 .04	135.2 9.1	85.6 10.8	313.7 21.2	2.0 .3	4.6 .8

[1] Based on average barometric pressure of 737.1 mm Hg.

[2] 1 unit = 5 mm pen deflection

[3] Changes in respiratory oxygen with constant carbon dioxide.

Table 3. Experimental values for variation of carbon dioxide in Study II.

CO_2 Changes[3]		Changes in parameters (mean ± S.E.)								
% CO_2	Ave.[1] P_{CO_2} mm Hg	P_{CO_2}	P_{O_2}	pH	Sys. Blood Pressure	Dias. Blood Pressure	Heart Rate	Resp. Period	Resp. Amplitude	Tracheal Pressure
		mm Hg	mm Hg		mm Hg	mm Hg	beats/min	cycles/sec	units[2]	cm H_2O
12	80.8	81.2±4.4	94.4±2.2	7.26±.10	156.8± 8.3	115.8± 5.8	283.9±14.7	3.7± .8	3.6±.3	70.6± 9.0
11	74.1	71.1 7.3	93.9 2.3	7.26 .05	140.2 5.7	112.1 9.7	281.0 9.3	3.5 .2	3.1 .3	68.9 9.0
10	67.3	70.2 3.4	94.1 1.5	7.27 .04	155.2 10.4	117.6 6.8	266.5 13.1	3.7 .7	3.1 .3	71.2 8.5
9	60.6	64.3 3.7	93.2 1.8	7.31 .07	158.2 10.0	120.2 6.9	260.4 12.0	3.7 .4	2.8 .2	73.3 7.7
8	53.9	56.1 3.0	92.9 1.1	7.32 .03	162.4 10.4	114.5 5.3	258.4 13.0	3.9 .2	2.6 .4	74.0 6.9
7	47.1	49.6 3.0	89.1 2.2	7.37 .03	166.3 11.1	122.5 7.8	259.7 11.9	4.3 .5	2.3 .1	82.7 8.5
6	40.4	44.5 2.8	89.3 2.1	7.39 .03	166.6 11.3	119.6 6.6	261.7 11.8	5.2 .8	1.8 .2	95.8 6.6
5	33.7	36.7 2.5	86.3 2.5	7.42 .03	163.6 11.2	120.0 8.1	260.1 12.6	3.3 1.0	1.1 .3	89.9 5.2
4	25.9	30.8 2.0	84.2 2.7	7.46 .03	157.1 11.4	116.6 8.0	244.7 16.8	2.3 1.4	.3 .2	96.0 5.4
3	20.2	25.0 1.8	81.8 3.1	7.50 .02	151.0 9.5	111.9 7.2	240.9 14.8	.2 .2	0.0 .0	98.3 6.8
2	13.4	18.2 1.6	77.4 4.2	7.58 .03	145.3 9.7	105.4 8.2	235.6 11.9	.2 .2	0.0 .0	93.8 7.8
3	20.2	24.4 3.4	79.0 2.2	7.51 .03	151.3 12.3	111.9 7.7	243.9 13.4	2.0 1.1	.1 .1	88.2 6.3
4	25.9	28.6 1.7	83.7 2.1	7.46 .02	156.0 10.1	115.5 7.2	261.9 14.6	2.7 1.0	.2 .1	88.9 6.5
5	33.7	33.8 2.2	85.8 1.9	7.41 .03	162.1 6.7	119.6 4.4	277.8 8.7	4.8 1.0	1.0 .2	90.1 10.0
6	40.4	40.3 2.5	87.0 2.1	7.37 .02	164.6 6.1	120.3 4.0	271.4 7.5	3.9 .5	1.8 .3	79.7 9.1
7	47.1	45.3 3.0	87.0 1.9	7.33 .02	167.9 5.5	121.4 3.4	265.9 8.9	3.4 .5	2.1 .3	79.1 10.1
8	53.9	53.1 3.9	88.3 2.1	7.31 .02	167.0 8.5	116.2 4.9	260.7 14.1	3.6 .4	2.5 .3	77.4 5.4
9	60.6	58.7 3.7	90.3 2.3	7.26 .03	165.3 8.8	114.8 4.9	257.1 13.4	3.5 .4	2.7 .3	70.5 7.0
10	67.3	63.7 4.3	90.7 2.5	7.24 .03	169.1 8.8	113.6 4.6	255.0 14.9	3.2 .4	2.9 .3	76.8 10.5
11	74.1	68.3 4.6	91.6 2.2	7.22 .03	160.4 7.9	108.3 10.1	261.0 11.4	2.8 .3	3.1 .3	74.0 11.2
12	80.8	72.1 3.2	91.1 1.8	7.20 .03	160.0 6.6	108.0 5.0	254.1 11.1	3.2 1.4	3.1 .3	72.7 5.9

[1] Based on average barometric pressure of 728.5 mm Hg.

[2] 1 unit = 5 mm pen deflection

[3] Changes in respiratory carbon dioxide with constant oxygen.

Table 4. Experimental values for variation of oxygen in Study II. (Trial 1).

%O_2 Changes[3]		Changes in parameters (mean ± S.E.)								
% O_2	Ave.,[1] P_{O_2} mm Hg	P_{CO_2}	P_{O_2}	pH	Sys. Blood Pressure	Dias. Blood Pressure	Heart Rate	Resp. Period	Resp. Amplitude	Tracheal Pressure
		mm Hg	mm Hg		mm Hg	mm Hg	beats/min	cycles/sec	units[2]	cm H_2O
50	336.8	36.9±3.0	234.3± 8.7	7.38±.00	160.5± 8.5	116.2± 5.7	242.5±16.4	5.4±1.1	1.0±.3	79.9±10.2
46	309.9	35.0 2.7	217.7 8.0	7.39 .04	164.3 8.1	118.4 5.8	237.3 13.2	4.9 1.1	1.1 .3	82.7 10.0
42	282.9	33.5 2.9	196.9 8.6	7.35 .04	163.2 8.6	118.5 5.5	237.8 17.3	5.4 1.6	1.2 .3	79.6 9.9
38	256.0	33.0 2.9	174.8 7.8	7.40 .04	163.9 8.0	119.0 5.4	238.4 17.4	5.2 0.9	1.2 .3	82.1 3.6
34	229.0	32.7 3.1	162.8 13.7	7.40 .04	164.8 9.9	119.5 5.3	243.4 18.4	6.5 1.0	1.3 .3	79.4 11.3
30	202.1	32.7 2.8	134.9 5.4	7.38 .04	165.4 7.8	120.3 5.6	243.2 17.3	5.5 1.9	1.5 .3	82.6 5.4
28	108.6	32.7 2.9	119.7 14.2	7.38 .04	166.8 8.4	121.5 5.5	248.2 17.3	5.2 .7	1.7 .3	80.0 13.5
26	175.1	33.0 2.8	112.5 4.3	7.37 .04	167.4 7.6	120.8 5.0	248.7 18.5	5.2 .7	2.0 .4	78.7 9.0
24	161.7	32.6 3.5	105.2 5.1	7.36 .04	166.5 8.6	120.5 5.7	249.1 19.6	5.0 .7	1.9 .4	81.2 11.6
22	148.2	32.8 2.7	98.8 4.1	7.36 .04	164.3 8.4	119.1 5.9	249.0 19.5	5.0 .7	2.0 .4	78.0 10.0
20	134.7	33.7 2.7	92.5 2.3	7.35 .04	160.2 8.4	115.6 5.4	250.7 20.3	4.6 .7	2.1 .4	70.0 11.1
18	121.2	33.0 2.6	83.9 2.1	7.36 .04	163.6 7.6	118.9 5.5	255.4 18.3	4.7 .5	1.9 .4	79.4 12.8
16	107.8	34.2 3.0	76.7 2.2	7.36 .04	162.7 8.0	117.0 5.2	259.0 16.4	4.3 .5	2.1 .3	76.3 7.1
14	94.3	33.7 2.6	67.4 15.7	7.36 .04	161.9 7.8	117.9	262.9 16.5	4.1 .4	2.2 .3	78.5 15.0
12	80.8	32.8 2.3	56.9 2.4	7.37 .04	161.6 8.3	117.5 5.6	273.2 4.4	4.0 .1	2.3 .3	75.0 10.3
10	67.4	34.0 2.5	41.5 2.9	7.36 .04	155.0 7.2	107.5 11.3	288.8 13.4	3.2 .4	2.7 .3	71.1 9.5
8	53.9	34.6 2.7	36.2 2.7	7.35 .05	150.2 7.6	100.3 5.1	306.4 10.9	2.9 .6	2.7 .4	71.1 11.8
6	40.4	37.3 7.0	31.3 4.3	7.38 .06	150.0 11.3	94.5 5.0	310.7 8.0	2.3 .2	3.3 .3	65.6 13.8

[1] Based on average barometric pressure of 728.5 mm Hg.

[2] 1 unit = 5mm pen deflection

[3] Changes in respiratory oxygen with constant carbon dioxide.

Table 5. Experimental values for variation of oxygen in Study II. (Trial 2).

% O$_2$ Changes[3]		Changes in parameters (mean ± S.E.)								
% O$_2$	Ave.[1] PO$_2$ mm Hg	P$_{CO_2}$	P$_{O_2}$	pH	Sys. Blood Pressure	Dias. Blood Pressure	Heart. Rate	Resp. Period	Resp. Amplitude	Tracheal Pressure
		mm Hg	mm Hg		mm Hg	mm Hg	beats/min	cycles/sec	units[2]	cm H$_2$O
50	336.8	34.8± 2.0	221.6±7.2	7.36±.03	161.2± 7.2	116.1±3.9	239.2±13.7	5.1±.6	1.4± .8	68.1± 8.4
46	309.9	34.4 1.9	202.6 9.0	7.39 .03	161.6 6.7	117.5 3.1	240.6 13.9	5.3 .6	1.5 .9	73.0 8.1
42	282.9	34.8 1.8	182.1 7.9	7.38 .03	161.8 6.6	115.2 3.2	241.5 14.2	5.1 .4	1.6 .9	70.9 8.0
38	256.0	34.1 2.1	162.5 8.6	7.37 .03	159.4 6.2	116.4 3.5	245.5 15.6	4.9 .5	1.6 .9	71.3 9.2
34	229.0	33.0 1.8	139.7 6.8	7.39 .03	166.7 4.1	118.5 2.6	245.8 17.2	4.5 .4	1.8 1.2	70.7 8.4
30	202.1	32.3 1.6	129.9 5.4	7.39 .03	159.9 6.4	115.3 3.9	246.8 17.1	4.5 .4	1.9 1.0	67.2 8.0
28	108.6	34.1 1.7	116.9 5.3	7.39 .03	161.4 6.4	115.7 4.2	251.0 16.6	4.6 .6	2.0 .9	67.0 8.0
26	175.1	34.9 1.7	110.1 4.6	7.39 .03	157.2 6.2	112.8 4.6	255.9 17.4	4.4 .4	1.8 .8	65.2 8.1
24	161.7	34.9 2.0	105.4 4.1	7.39 .03	161.7 7.7	114.9 3.9	261.9 17.6	4.2 .4	2.0 1.0	61.3 8.2
22	148.2	34.0 1.7	95.9 3.9	7.39 .03	154.4 6.3	112.1 5.7	257.6 15.5	4.6 .4	2.0 .8	71.7 8.7
20	134.7	34.6 2.0	87.4 3.6	7.39 .03	155.8 8.2	110.4 4.9	258.7 28.0	4.7 .5	2.2 .9	76.4 9.5
18	121.2	34.6 1.8	78.9 2.9	7.39 .03	152.4 7.8	107.9 4.6	267.6 17.2	4.8 .6	2.4 1.0	69.4 9.0
16	107.8	35.5 2.0	71.7 2.5	7.39 .03	151.2 7.0	106.0 4.0	269.3 17.9	4.0 .4	2.4 1.0	65.5 9.1
14	94.3	35.0 1.7	65.0 2.8	7.38 .03	153.6 7.3	106.0 4.2	268.1 17.0	4.0 .4	2.6 1.1	62.8 8.9
12	80.8	33.5 1.7	59.9 1.9	7.38 .03	147.1 6.6	102.8 4.3	282.2 13.8	3.5 .3	2.6 .8	75.1 12.4
10	67.4	33.9 1.6	43.2 1.8	7.37 .03	149.1 6.8	98.3 4.6	293.2 16.8	3.1 .4	3.0 1.2	70.3 8.3
8	53.9	35.6 1.6	33.7 1.7	7.33 .03	143.4 7.1	96.6 3.4	314.9 18.9	2.4 .2	3.4 1.9	76.3 12.7
6	40.4	34.1 3.2	27.6 1.5	7.34 .02	157.8 9.0	105.7 5.6	305.3 27.6	1.8 .2	4.2 1.1	71.8 6.7
4	26.5	34.5 18.2	18.0 1.1	7.30 .06	225.7 15.1	124.6 7.7	207.0 12.0	2.4 .4	5.2 2.8	55.7 15.0

[1] Based on average barometric pressure of 728.5 mm Hg.

[2] 1 unit = 5 mm pen deflection

[3] Changes in respiratory oxygen with constant carbon dioxide.

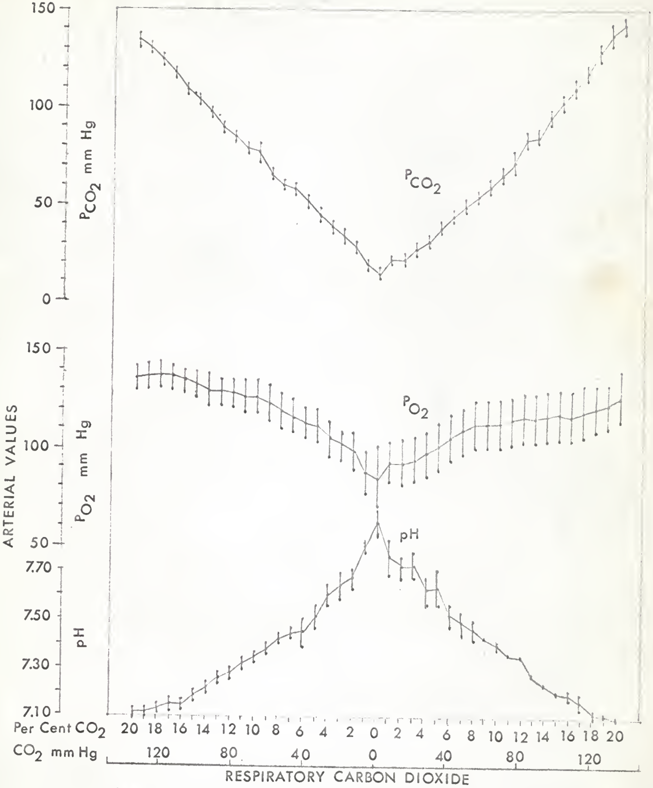

Fig. 5. Study I, Part A. Effect of variation in respired P_{CO_2} on arterial P_{CO_2}, P_{O_2}, and pH. Although the P_{O_2} of the gas mixture was constant, at 18% O_2 (121 mm Hg P_{O_2}), the arterial P_{O_2} was found to decrease as P_{CO_2} decreased.

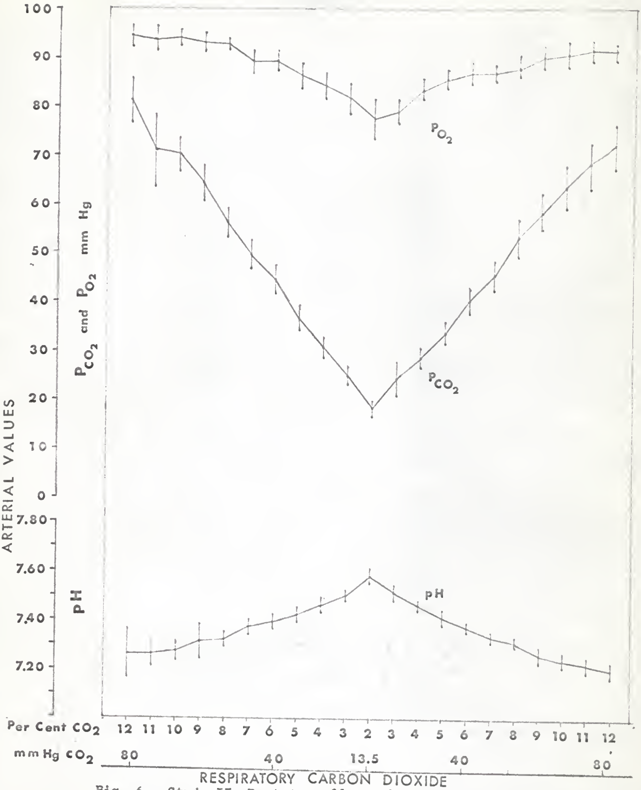

Fig. 6. Study II, Part A. Effect of variation in respired P_{CO_2} on arterial PCO_2, PO_2, and pH. Constant PO_2 in gas mixture (20% O_2) = 133 mm Hg.

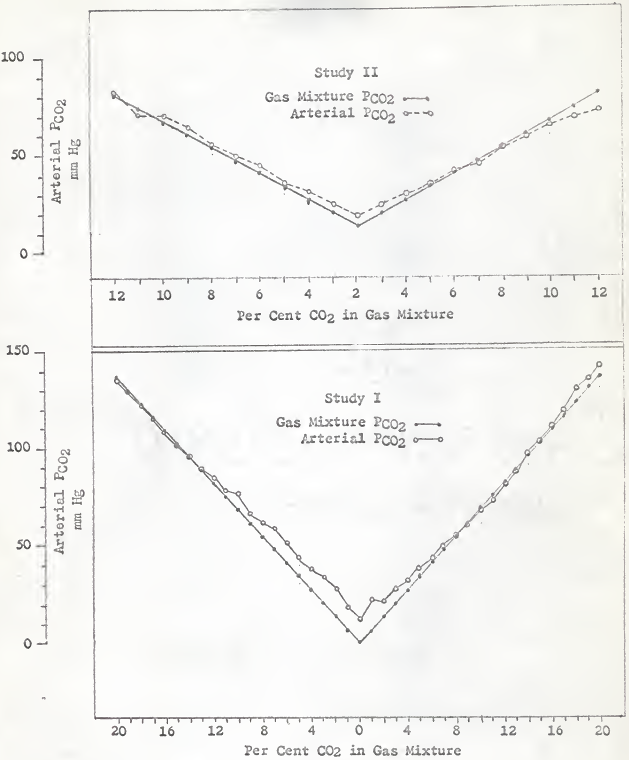

Fig. 7. Comparison of P_{CO_2} in gas mixtures with obtained arterial P_{CO_2}. The arterial P_{CO_2} was greater than the gas mixture P_{CO_2} at the lower carbon dioxide tensions. The difference was greater in Study I than in Study II.

Therefore, a lower P_{CO_2} reading might have been obtained if a longer time had been allowed before taking the reading. Second, the bird is producing a certain amount of CO_2 as a result of metabolic processes. This endogenous carbon dioxide may account for part of the difference between the P_{CO_2} of the gas mixture and the arterial P_{CO_2}. However, it would also be expected that endogenous carbon dioxide would give an increased arterial P_{CO_2} at higher gas mixture P_{CO_2} levels. But, since the P_{CO_2} electrode responds logarithmically and the pen deflection is much less for a unit P_{CO_2} at higher levels, the small difference between gas mixture P_{CO_2} and arterial P_{CO_2} probably can not be detected.

There is a third possible explanation for the difference between arterial P_{CO_2} and gas mixture P_{CO_2} at low levels being greater in Study I than in Study II. This stems from the fact that the initial carbon dioxide tensions in the experiment were higher in Study I (20%) than in Study II (12%). Thus, there could be a greater amount of combined carbon dioxide in the blood due to buffering of the initial high levels, which would be released at the lower carbon dioxide tensions. That the amount of combined carbon dioxide was higher in Study I than in Study II can be determined from the experimental pH determinations. The pH is related to the ratio of combined CO_2 to free CO_2 in the plasma by the equation: $pH = pK' + \log \frac{\text{combined } CO_2}{\text{free } CO_2}$. Helbacka _et al_. (1964) have determined the relationship of pK' to pH in the chicken. The pK' increases as the pH decreases. The ratio of combined to free CO_2 for selected percentages of carbon dioxide is compared for Study I and Study II in Table 6.

Table 6. Ratio of combined CO_2 to free CO_2 with variation in P_{CO_2} compared for Study I and Study II. Values for pK' from Helbacka et al., (1964).

	Study I				Study II				
Per Cent CO_2	Diff.[1]	Average pH	pK'	$\frac{\text{combined } CO_2}{\text{free } CO_2}$	Per Cent CO_2	Diff.[1]	Average pH	pK'	$\frac{\text{comb. } CO_2}{\text{free } CO_2}$
12	3.1	7.28	6.10	15:1	12	0.4	7.26	6.10	15:1
6	10.4	7.45	6.09	20:1	6	4.1	7.39	6.09	18:1
2	13.8	7.68	6.08	40:1	2	4.8	7.58	6.08	30:1

[1] Arterial P_{CO_2} — respiratory gas P_{CO_2}

From this table it can be seen that the amount of combined CO_2 to be eliminated at low carbon dioxide levels was greater in Study I than in Study II. Thus, the arterial P_{CO_2} at low carbon dioxide levels seems to have been influenced by the previous experimental conditions.

The changes which occurred in pH with variation in P_{CO_2} also tend to show that there may have been more combined CO_2 involved in Study I than in Study II. The pH values were generally higher in Study I when compared with those in Study II. In general, both studies showed a decrease in values for pH with increased respiratory P_{CO_2}. The average of pH values obtained at levels of 5% CO_2 with 18-20% O_2 was 7.46, when combining both studies.

Although the P_{O_2} of the gas mixture was held constant during the changes in P_{CO_2}, the arterial P_{O_2} showed definite variation (Figs. 5 and 6). The arterial P_{O_2} decreased as the arterial P_{CO_2} decreased. The arterial P_{O_2} decreased from about 130 mm. Hg at 20% CO_2 (136 mm. Hg. P_{CO_2}) to 120 mm. Hg at 12% CO_2 (82 mm. Hg P_{CO_2}) and to 85 mm. Hg at 0% CO_2 in Study I. In Study II the range was from about 90 mm. Hg P_{O_2} at 12% CO_2 (81 mm. Hg P_{CO_2}) to 77 mm. Hg at 2% CO_2 (13 mm. Hg P_{CO_2}). The arterial P_{O_2} during variation in P_{CO_2} was generally higher in Study I than in Study II. The occurrence of changes in P_{O_2} during variation in P_{CO_2} will be discussed in connection with tracheal pressure changes.

Effect of Variation in Respiratory P_{O_2} with Constant P_{CO_2} on Arterial P_{O_2}, P_{CO_2}, and pH

The effect of variation in oxygen tension of the respiratory gas mixture on arterial P_{CO_2}, P_{O_2}, and pH is shown in Figs. 8, 9, and 10. The variability of arterial P_{O_2} tended to increase with the increase in P_{O_2}.

Although arterial P_{O_2} increased with the increase in respiratory oxygen tensions, the divergence between the two is notable. The upper part of Fig. 11 presents a direct comparision of the P_{O_2} of the gas mixture and the obtained arterial P_{O_2}. The average P_{O_2} of the gas mixtures for Study I and Study II are slightly different due to the different average barometric pressures, 737.1 mm. Hg and 728.5 mm. Hg respectively. The difference between the P_{O_2} of the gas mixture and the arterial blood increases as the P_{O_2} is increased. At 50% O_2 the difference was about 100 mm. Hg. At low oxygen tensions the difference was smaller. In Study II the arterial P_{O_2} was lower than expected at the low oxygen tensions; while in Study I the arterial P_{O_2} was higher than expected. The arterial values for P_{O_2} in Study I were comparatively higher than in Study II, especially at levels below 30% O_2.

The lower part of Fig. 11 shows directly the differences between P_{O_2} in the gas mixture and the arterial P_{O_2}. The differences are expressed as the gas mixture P_{O_2} – arterial blood P_{O_2}. Thus, a negative value was obtained for the arterial P_{O_2} values below 18% in Study I. The theoretical difference curve

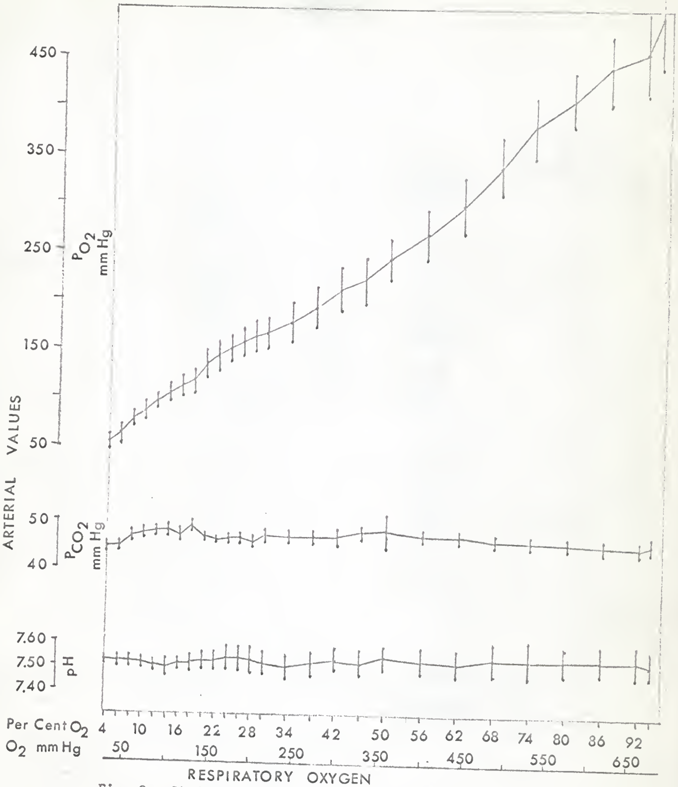

Fig. 8. Study I, Part B. Effect of variation in respired P_{O_2} on arterial P_{O_2}, P_{CO_2}, and pH. Constant P_{CO_2} in gas mixture = 34 mm Hg. P_{O_2} was increased from low values to high values.

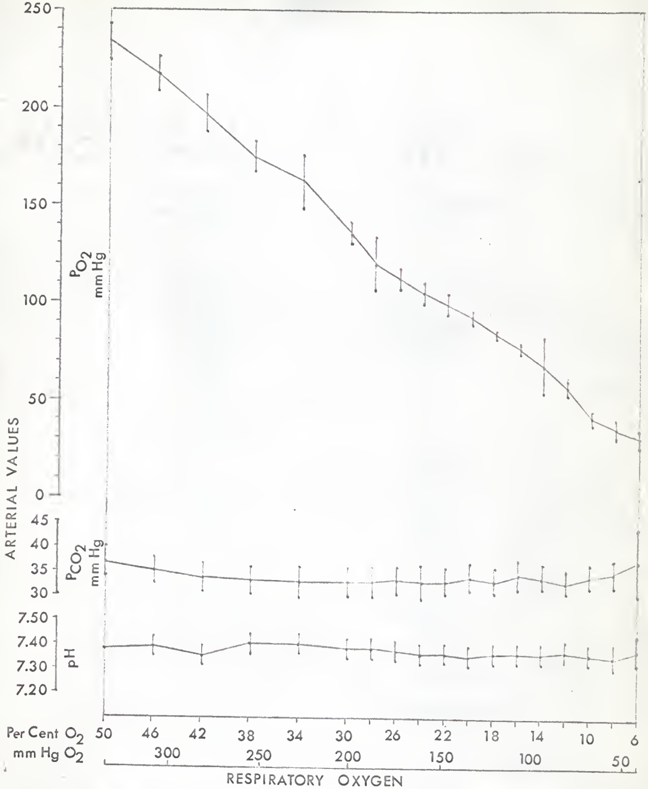

Fig. 9. Study II, Part B (Trial 1). Effect of variation in respired P_{O_2} on arterial P_{O_2}, P_{CO_2}, and pH. Constant P_{O_2} in gas mixture = 33 mm Hg. P_{O_2} was lowered from high levels to low levels.

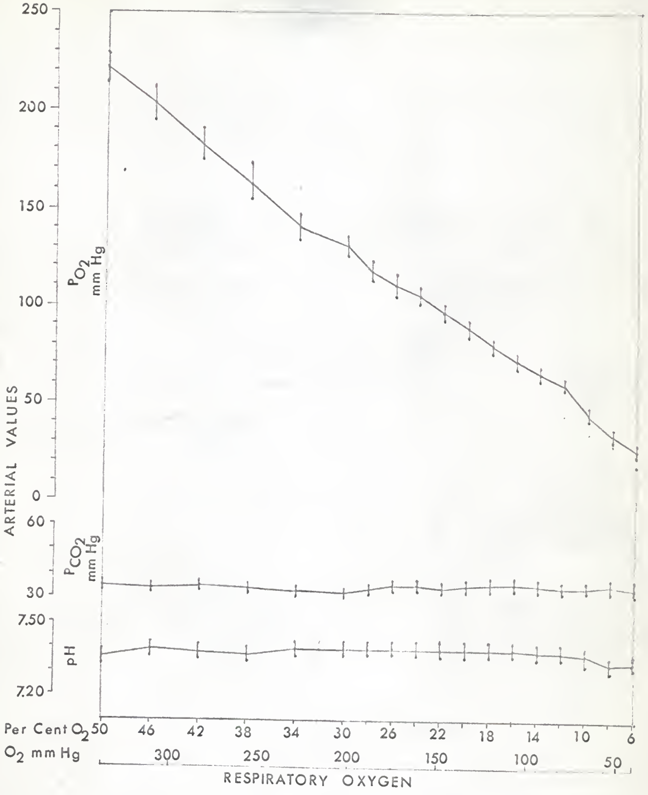

Fig. 10. Study II, Part B (Trial 2). Effect of variation in respired P_{O_2} on arterial P_{O_2}, P_{CO_2}, and pH. Constant P_{CO_2} in gas mixture = 33 mm Hg. P_{O_2} was lowered from high levels to low levels.

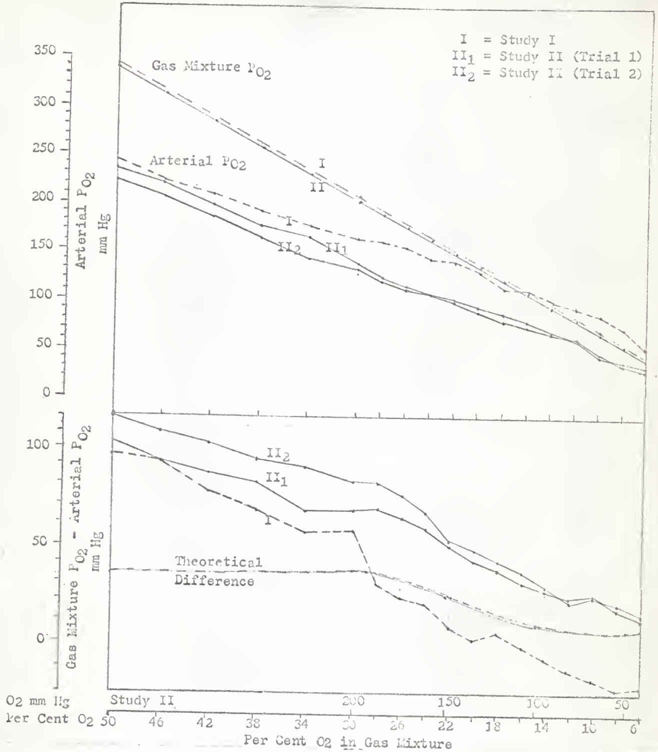

Fig. 11. Comparison of P_{O_2} in gas mixture and in arterial blood and their differences with per cent O_2 in gas mixture. Upper part: Comparison of the respiratory and the arterial P_{O_2} with the per cent O_2 in the gas mixture. Lower part: Comparison of differences between gas mixture and arterial P_{O_2} with per cent O_2 in gas mixture. See text for full explanation for theoretical curve.

is adapted from Rahn and Fahri (1964). This curve represents the sum of the effects of the three factors, which have been used by investigators in explaining the differences found between arterial P_{O_2} and alveolar P_{O_2}. They are (1) the diffusion factor, (2) the ventilation-perfusion factor, and (3) the venous admixture factor. Differences due to the diffusion and ventilation-perfusion factor are usually not large except in disease. The venous admixture factor, however, theoretically increases with increasing P_{O_2}. The addition of a small quantity of venous blood from the bronchial circulation and from shuts within the lung to the oxygenated blood in the pulmonary vein has a greater effect in reducing P_{O_2} at the higher oxygen tensions due to the flat shape of the oxyhemoglobin dissociation curve at these higher oxygen tensions (Rossier _et al._, 1960). The air capillary P_{O_2} - arterial P_{O_2} differences in Study I and Study II at the higher oxygen tensions were even greater than might be expected from this theoretical curve and appear to be only partly explained by these factors (Fig. 11), assuming that they may be applied to the chicken. It is possible that the nucleated red blood cells of the chicken utilize enough oxygen to decrease the arterial oxygen tension somewhat. However, a more plausible explanation for the difference in oxygen tensions involves the measurement of the oxygen tension with the P_{O_2} electrode. Very recent work by Dr. Richard Boster in this laboratory (unpublished data) shows that the electrodes do not produce the same magnitude of current change and hence the same magnitude of recorder pen deflection when calibrated with gases at a given tension as when calibrated with liquids equilibrated with gases at this tension. The equilibrated liquid will yield about 20-25% less electrode response than the gas at high gas P_{O_2} values (500-550 mm. Hg). If the venous admixture factor and the electrode calibration factor are taken into account, the measured arterial P_{O_2} is very comparable to the P_{O_2} in the respiratory gas.

The respiratory carbon dioxide tensions were kept constant during variation in oxygen tensions. In general, there tended to be little change in arterial pH and in arterial P_{CO_2} with changes in oxygen tension (Figs. 8, 9, and 10). The arterial P_{CO_2} was slightly reduced at the beginning in Study I (Fig. 8) with the initially low P_{O_2}. This lowering of P_{CO_2} was not noticed in Study II, where the low P_{O_2} levels were approached gradually. The valuos for pH and P_{CO_2} in Study I were slightly higher than in Study II. Also the P_{CO_2} of the arterial blood in Study I (Fig. 8) was about 10 mm. Hg higher than in the gas mixture. Arterial P_{CO_2} was quite close to the P_{CO_2} of the gas mixture in Study II (Fig. 9 and 10). A possible explanation for the higher pH and P_{CO_2} in Study I may be because the combined carbon dioxide was higher in Study I at the lower carbon dioxide levels. The study of variation in P_{CO_2} (Fig. 5) preceded the study of variation in P_{O_2} in the experimental procedure. The higher pH values obtained in the later oxygen studies indicate that there was still excess combined CO_2. In the presence of normal carbon dioxide tensions in the lungs, the excess combined CO_2 would be released and then the arterial P_{CO_2} would be higher than expected.

Effect of Variation in Carbon Dioxide Tensions on Blood Pressure, Heart Rate, Respiratory Period, Respiratory Amplitude, and Tracheal Pressure

Blood Pressure and Heart Rate. Blood pressure and heart rate decreased with decreasing carbon dioxide tensions (Figs. 12 and 13). This decrease was more apparent in Study I (Fig. 12), in which the respired carbon dioxide was decreased to 0%. The greatest change in blood pressure occurred at about the same carbon dioxide tensions at which respiratory movements ceased.

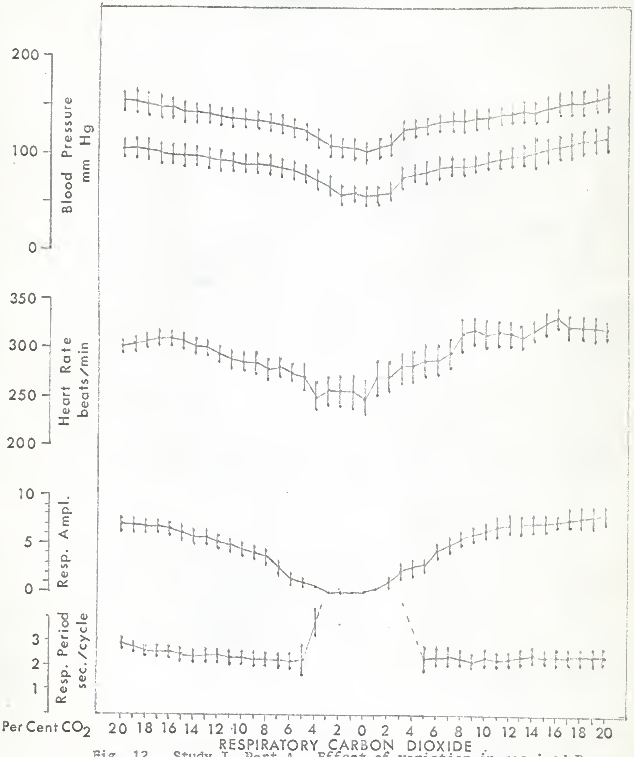

Fig. 12. Study I, Part A. Effect of variation in respired P_{CO_2} on blood pressure, heart rate, respiratory amplitude, and respiratory period.

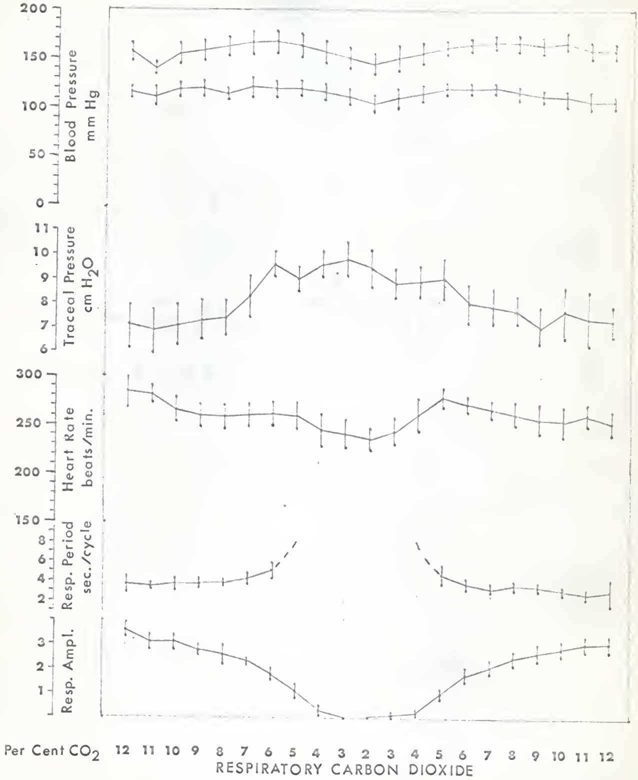

Fig. 13. Study II, Part A. Effect of variation in respired P_{CO_2} on blood pressure, tracheal pressure, heart rate, respiratory period, and respiratory amplitude.

Heart rate was generally higher during changes in P_{CO_2} in Study I than in Study II (Figs. 12 and 13). This higher heart rate might possibly have been due to the higher concentration of carbon dioxide, since carbon dioxide seems to have a stimulating effect on the heart (Dukes, 1955). Heart rate also tended to be higher after respiratory movements were resumed than before they ceased.

Variation in oxygen tensions did not produce any appreciable changes in blood pressure and heart rate except at low oxygen tensions (Figs. 14, 15, and 16). In Study I (Fig. 14), there was a slight increase in heart rate during hypoxic conditions; while in Study II (Figs. 15 and 16), the heart rate showed a definite increase, starting at about 14% O_2, to around 50 beats/min at 8% O_2. Heart rate in the mammal is also generally found to increase during hypoxic conditions (Korner, 1959).

Blood pressure during hypoxia tended to decrease and show an increased pulse pressure (Figs. 14, 15, and 16). Carrier et al. (1964) have shown that the local effect of hypoxia on isolated vessels is vasodilation. While an increase in only heart rate or an increase in only cardiac output would tend to increase blood pressure, a decrease in peripheral resistance would tend to decrease blood pressure and an increase in viscosity (which may occur during low oxygen or high carbon dioxide) will tend to produce dilation of vessels (Best and Taylor, 1961). It would appear that the decrease in blood pressure which occurred during hypoxia indicates the presence of peripheral vasodilation.

The average blood pressure of the birds was lower in Study I than in Study II during the variation in oxygen tension (Figs. 14, 15, and 16). This may have been due to vasodilation produced by the initial hypoxia in Study I.

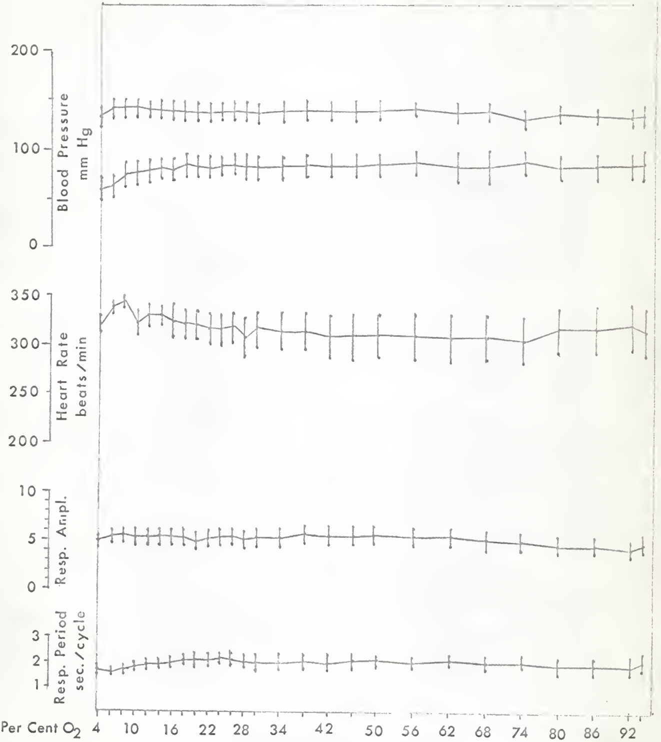

Fig. 14. Study I, Part B. Effect of variation in respired P_{O_2} on blood pressure, heart rate, respiratory amplitude, and respiratory period.

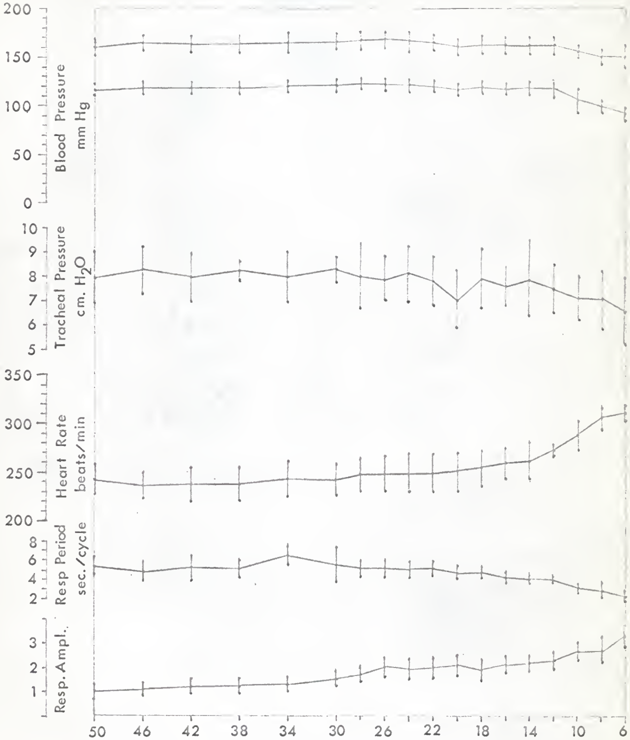

Fig. 15. Study II, Part B (Trial 1). Effect of variation in respired PO_2 on blood pressure, tracheal pressure, heart rate, respiratory period, and respiratory amplitude.

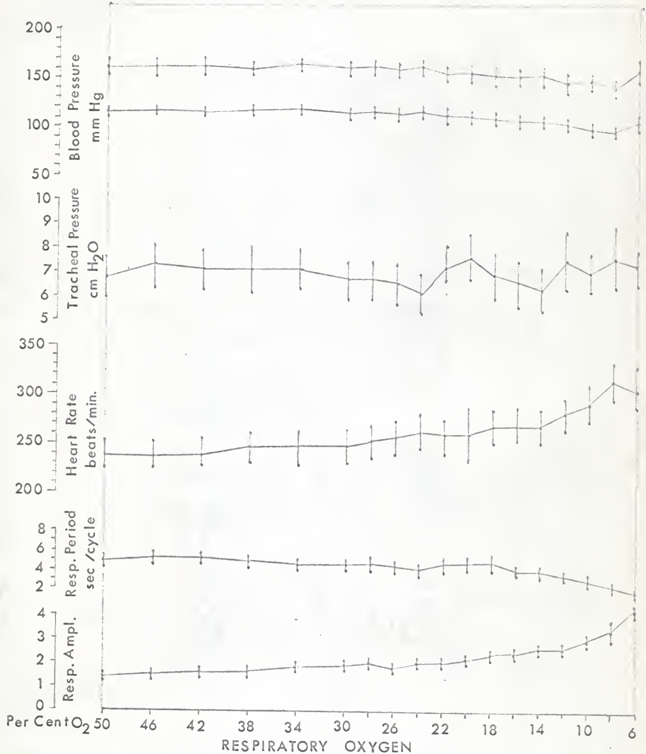

Fig. 16. Study II, Part B (Trial 2). Effect of variation in respired PO_2 on blood pressure, tracheal pressure, heart rate, respiratory period, and respiratory amplitude.

Respiratory period and Respiratory Amplitude. As carbon dioxide tensions decreased in Study I and Study II, the respiratory period (seconds/respiration) increased and the respiratory amplitude decreased (Figs. 12 and 13). Respiratory movements usually ceased at around 4% CO_2. A few birds showed slight respiratory movements at lower percentages of CO_2, which accounts for the fact that these averages are not quite zero. Respiratory amplitude was measured in terms of mm. of pen deflection with the recorder at a constant gain setting. Zero drift occurred between experiments so that direct comparisons between birds could be made. The magnitude of the pen deflection was a direct reflection of the magnitude of sternal movements. The data were transformed for presentation in the Figures so that 5 mm. pen deflection is equal to 1 unit. The respiratory amplitude increased with increasing carbon dioxide levels (Fig. 12 and 13). It has been shown for mammals that there is an increased ventilatory response to carbon dioxide up to levels of 12%. Above these levels, the respiratory response decreases and carbon dioxide tends to act more and more as an anesthetic (Dripps and Comroe, 1947). A decrease in respiratory response to carbon dioxide levels above 12% was not found to occur in the chicken. Respiratory amplitude tended to level off at about 14% CO_2. Furthermore, changes in tension at the higher levels of carbon dioxide tended to result in an excitatory motor response in the chicken even though the level of anesthesia was adequate at the lower CO_2 levels. These data indicate that these high levels of CO_2 do not produce as much central depression in the chicken as in mammals. The leveling off of respiratory amplitude at these high CO_2 levels may indicate that the bird was simply not physically capable of breathing any deeper and that maximum respiratory movements were produced by levels of about 14% respired CO_2.

As P_{CO_2} was increased, there was little change in respiratory period except at low carbon dioxide tensions (Figs. 12 and 13). The respiratory period was greatly lengthened before respiratory movements ceased at low CO_2 tensions, with the respiratory period (sec./cycle) approaching infinity. The respiratory period tended to level off at about 5% CO_2 in Study I and about 7% in Study II. Thus, the higher levels of respired CO_2 did not act to decrease respiratory period or conversely to increase respiratory rate.

With decreasing oxygen tensions during Study II, the respiratory amplitude gradually increased and the respiratory period gradually decreased (Figs. 15 and 16). During oxygen changes in Study I, respiratory amplitude showed little change, but respiratory period was slightly decreased during hypoxic conditions (Fig. 14). However, the respiratory amplitude was higher in Study I than in Study II. Since increases in respired P_{CO_2} act primarily to increase respiratory amplitude and since the concentration of carbon dioxide was higher during the variation of oxygen in Study I than in Study II, the generally higher amplitude in Study I may have been due to the elevated amount of carbon dioxide present.

Tracheal Pressure. Tracheal pressure, measured in Study II, showed an increase at low carbon dioxide levels (Fig. 13). The greatest change in tracheal pressure occurred when respired carbon dioxide was below 8%.

Unfortunately a simple measurement of tracheal pressure does not differentiate between the possible causes of changes in this variable. An increase in tracheal pressure could be due to bronchoconstriction or due to changes in pulmonary vasculature. Other investigators have found that the increase in tracheal pressure is produced by changes in the air passageways in the lung. An increase in airway resistance was found in the dog (anesthetized with pentobarbital) under conditions of decreased carbon dioxide levels in the blood (Severinghaus et al., 1961). This effect was reversed with 6% CO_2, isoproterenol, or 100% N_2. It has been reported that inhalation of 10-12% O_2 or 5 to 8% CO_2, or stimulation of the carotid body with 2-10 µmg. of nicotine will increase total lung resistance under chloralose-urethane anesthesia (Nadel and Widdecombe, 1961). Forester (1964) has found that an increase in alveolar P_{CO_2} increases the ease with which carbon monoxide reaches the red blood cell in the lung. This indicates an increase in diffusion of oxygen from alveoli to red blood cell under the influence of high P_{CO_2}.

Although the respiratory movements of the birds could not alter the quantity of gas which passed through the lungs under these artificial ventilation conditions, an increase in tracheal pressure could indicate a reduction in flow past the gas exchange surfaces of the lungs. The parabronchi have an abundance of smooth muscle cells which surround them in a unique network. A slight contraction of these muscle cells would decrease the gas flow through the parabronchi and hence over the gas exchange surfaces and would act to shunt the gas through the direct bronchial pathways to the air sacs. Such a shunt would have the effect of diminishing the exchange, especially oxygen, between gases and blood. Such an explanation also seems supported by the reduced arterial P_{O_2} which occurred during increased tracheal pressure at low respired carbon dioxide levels (Figs. 5 and 6).

No significant change in tracheal pressure was found with the variation of oxygen tension (Figs. 15 and 16). The tracheal pressure means were, however, more erratic when below 22% O_2. There was little change in tracheal pressure at higher oxygen tensions. Some birds tended, however, to show definite gradual decreases in tracheal pressure with hypoxia.

Control of variation in tracheal pressure probably is necessary in the control of blood gas levels. It appears that further evaluation of the influence of gas tensions on airway resistance will require consideration of at least three factors. First, changes in tracheal pressure do occur with changes in carbon dioxide and possibly may be influenced by low levels of respired oxygen. Each of these gases should be studied over a range of tensions. According to our results, the 5-8% CO_2 level, which was used by Nadel and Widdecombe (1961), is in a region in which the tracheal pressure is changing. It may be that there is a threshold in the effect of carbon dioxide on tracheal pressure. Second, the duration of hypoxia may be a factor influencing tracheal pressure. Some variation has been noted in experimental responses of tracheal pressure to hypoxia. In the present experiment the oxygen level in the respired gas was reduced to 4% (not reported on the graphs) in two birds with a concomitant marked drop in a tracheal pressure occurring. It may be that prolonging hypoxia produces a drop in tracheal pressure. Third, the type of anesthetic used may be important. Tracheal pressure responses to hypoxia have been obtained with chloralose or chloralose-urethan rather than with barbiturates (Widdecombe, 1963).

SUMMARY

A method has been studied by which the arterial blood gas tensions in the chicken can be controlled with the use of the unidirectional artificial respirator. Simultaneous determinations have been made for the arterial P_{O_2}, arterial P_{CO_2}, arterial pH, blood pressure, heart rate, tracheal pressure, respiratory period, and respiratory amplitude which correspond to given respiratory gas mixtures. The respiratory gas mixtures ranged in steps (1) from 20-0-20% CO_2 and from 4-94% O_2 in Study I and (2) from 12-2-12% CO_2 and from 50-6% O_2 in Study II.

The arterial carbon dioxide tensions tended to coincide with the respiratory gas mixture tensions except at low levels of carbon dioxide. This difference may have been due to a build-up of combined carbon dioxide from the preceding exposure to high CO_2 or from metabolically produced carbon dioxide. The difference between the arterial oxygen tension and the respiratory oxygen tension tended to increase with increasing P_{O_2}. This difference seemed to be due mainly to the increased effect of venous admixture at high oxygen levels and to the use of gases rather than liquids for calibrating the oxygen electrode.

Measurements of respiratory movements indicated that the respiratory response does not decrease at high levels of carbon dioxide in the chicken. The increase in tracheal pressure, which occurred at low carbon dioxide levels, may be associated with the decreased arterial oxygen tensions which occur at low carbon dioxide levels. An increase in heart rate and a decrease in blood pressure were found to occur at the lower levels of oxygen.

With the use of liquids rather than gases for oxygen electrode calibrations and taking possible effects of combined carbon dioxide and venous admixture into consideration, unidirectional respiration does make possible the control of arterial blood gas tensions in the chicken.

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PHYSIOLOGICAL EFFECTS OF VARIATION IN LEVELS OF RESPIRED CARBON DIOXIDE AND OXYGEN IN THE CHICKEN

by

PAULA JEAN RAY

B.A., Kansas State University, 1962

AN ABSTRACT OF A MASTER'S THESIS

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Manhattan, Kansas

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A method for the experimental control of changes in blood gas tensions *in vivo* has made possible the study of chemoreceptor responses over a range of blood gas tensions and the study of possible interaction of responses of the different chemoreceptors. The unidirectional respirator can be used in the chicken to produce desired changes in blood gas tensions. This study has been made to determine the relationship between arterial blood gas tensions and respiratory gas tensions when these tensions were widely varied. In conjunction with the continuous measurement of arterial P_{O_2}, P_{CO_2}, and pH, blood pressure, heart rate, tracheal pressure, respiratory period (inverse of rate), and respiratory amplitude were also measured on adult male chickens.

Study I included the simultaneous measurement of each of the above parameters except tracheal pressure under conditions in which the gas mixtures (BTPS) used with the unidirectional respirator were varied (1) from 20% CO_2 down to 0% CO_2 and back up to 20% CO_2, while holding oxygen at 18% and (2) from 4% O_2 up to 94% O_2, while holding carbon dioxide at 5%. Study II included measurement of the same parameters, with the addition of tracheal pressure. The gas mixtures in Study II were varied (1) from 12% CO_2 down to 2% CO_2 and back up to 12% CO_2 and (2) from 50% O_2 down to 6% O_2(two trials on each bird).

The Beckman modular cuvette, containing electrodes sensitive to P_{CO_2}, P_{O_2}, and pH, was used to make continuous measurements of these variables. Analyzed gases containing various percentages of O_2 and CO_2 were used to calibrate the P_{O_2} and P_{CO_2} electrodes. Appropriate transducers were used for the measurement of the other parameters. An Offner Type S multichannel pen recorder was used for simultaneous recording of the parameters. Constant temperature water baths were used with the modular cuvette and for warming and humidifying the gas mixtures in the unidirectional respirator. Body temperature was also monitored.

The results of the experiment showed close agreement between the P_{CO_2} of of the respiratory gas mixture and the arterial P_{CO_2}, except at low tensions of respired P_{CO_2}. The difference found in carbon dioxide tensions at low CO_2 may have been due to a build-up of combined CO_2, from preceding exposure of the blood to high P_{CO_2}. Also, since endogenous CO_2 is continuously produced, a small amount of carbon dioxide tension would be expected even at low respiratory values of P_{CO_2}.

The agreement between the P_{O_2} of the respiratory gas mixture and the arterial P_{O_2} was less exact. As the P_{O_2} was increased, the difference between the respired gas tension and arterial P_{O_2} increased. This difference could be explained by several factors, including venous admixture, use of gases rather than liquids for electrode calibrations, and ventilation-perfusion differences.

Findings from measurement of other parameters include lower blood pressure and higher tracheal pressure at low respired P_{CO_2}, and little variation of these parameters with changes of respired oxygen except at low O_2 tensions where there was an increase in heart rate and decrease in blood pressure. Alteration of respired carbon dioxide seemed to have little effect on respiratory period, but respiratory amplitude increased with increasing P_{CO_2}. Decreasing the oxygen tension increased amplitude and decreased period. Respiratory movements usually ceased when the respired CO_2 went below 4%.

The unidirectional respirator can be used to control blood gas tensions if the factors which cause differences between respiratory gas tensions and arterial blood gas tensions are taken into consideration.